PERICARDITIS: ITS SYMPTOMS AND DIAGNOSIS.[1]

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Pericarditis is, as a rule, more damaging to the heart than endocarditis. It is almost certain to shorten the life of the person afflicted unless an intercurrent disease should anticipate its effects.

Its causes, to which, merely for the sake of completeness, I would briefly refer, are in the main twofold: direct extension and hæmatogenous infection. Traumatic pericarditis is so rare that it may be disregarded.

The pericardium may be involved by direct extension from neighboring structures in the thorax, such as the lung, the pleura, the mediastinal and bronchial lymph glands, and the œsophagus; also from the endocardium or the myocardium, from the great vessels near their origin, and from aneurysms. Inflammatory processes below the diaphragm may likewise cause pericarditis by extension.

Hæmatogenous infection is the most frequent, and gives rise to the type of pericarditis accompanying acute articular rheumatism and other infectious diseases. A form of particular interest is that associated with Bright's disease and uræmia, the true nature of which is still in doubt. Unquestionably, however, its cause is hæmatogenous, and is either a toxin or a bacterium. It may develop in any of the forms of nephritis, but is most frequently encountered in acute Bright's disease, in the early stages of the large white kidney, and in chronic interstitial nephritis. Frederick T. Roberts says that it is almost never found in cases of typical large white kidney. In one of my cases, however, the kidneys were in an advanced stage of chronic parenchymatous nephritis (the so-called large white kidney); but, as the patient had pneumonia also, the share of the kidneys in producing the pericarditic process is not readily fixed.

Nephritic, or uræmic, pericarditis is nearly always of the fibrinous variety, with little fluid; but it may be serous or hemorrhagic. I am not forgetting simple hydropericardium, which is sometimes present in cases of chronic nephritis complicated with general dropsy and effusions into the serous cavities. I have, however, been surprised at the rarity with which hydropericardium occurs when the conditions supposed to favor it, renal and cardiac dropsy, are present.

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[1] Read before the College of Physicians of Philadelphia, May 4, 1904.

Pericarditis is quite common in cases of pneumonia, especially when this disease is on the left side. It is frequently overlooked, but should always be thought of as one of the possible causes of so-called delayed resolution, or, rather, delayed crisis. In my experience it has usually been purulent; but it may be serofibrinous. It is generally accompanied with a mediastinopericarditis or a pleuropericarditis of the same nature.

I must say a few words in regard to pericarditis of tuberculous origin. This is usually of the plastic, obliterating variety, but sometimes it is purulent. It is rare, and is commonly overlooked during life; and even after death its true nature may not be recognizable without a microscopic examination.

In taking up now the symptoms and diagnosis I shall not discuss these two aspects of the subject separately, but shall consider the principal features as they present themselves to the examiner.

1. THE FRICTION SOUND. This is most marked in acute fibrinous pericarditis, the commonest form. Its site of maximum intensity is to the left of the sternum in the third and fourth interspaces, and sometimes in the second. In rare instances, as in two under my observation at the present time, it is heard first or solely at the apex. It is usually of a churning character, superficial, and markedly intensified by pressure with the stethoscope. It may have a gallop-rhythm, and in pronounced cases it has a peculiar chugging quality, like the noise made by a locomotive just starting to move. If confined to the apex, it may closely resemble a presystolic murmur; while when loudest at the base it may simulate double aortic disease. The character of the pulse, the size of the heart, and the history, together with careful examination so as to discover the second aortic sound, will aid in the diagnosis from aortic-valve disease.

When the friction sound is loud it is often difficult to distinguish existing endocardial murmurs. These may be heard if a double stethoscope is used and no pressure is applied. Pressure brings out the friction sound and thereby renders the murmurs faint or inaudible. I have recently been able to study the heart before the development of pericarditis in two cases. One was that of a young Italian with aortic regurgitation. In the other case, also that of an Italian, there was mitral insufficiency. As the friction sound increased in loudness, the murmurs in both grew fainter and fainter, and at present the aortic murmur is inaudible; while the mitral systolic can be heard when the stethoscope is placed lightly upon the chest. In some cases a mitral murmur, obscured in the precordial area by the confused noise of the friction sound, may be heard in the axilla.

Pleural and pleuropericardial friction must also be differentiated from the ordinary pericardial friction. Both are modified by respiration.

The existence of a friction sound does not exclude the presence of fluid. In one of the cases in which Dr. Roberts tapped and obtained considerable fluid, the friction sound had not disappeared. The fluid is often held in compartments separated from one another by bands or areas of adhesion, which preserve the friction sound. In some cases the friction sound persists at the apex after it has disappeared elsewhere, probably because the greater movability of the apex retards the formation of adhesions.

Friction may cause a palpable thrill. Of my recently observed cases this thrill was present in two out of nine. The loudest friction may, however, be entirely unaccompanied with thrill.

2. Pain and Tenderness. These were present in four of the nine cases. In these four the pain was intense. In three the tenderness was an exquisite hyperæsthesia, so that percussion became torture. In one of the cases the pain was paroxysmal, and was apparently as severe as in angina pectoris, although its direction of radiation was different. It started at the wrists and ran with great violence up the arm and across the shoulders into the chest. In another case there was moderate pain and tenderness in the epigastrium, and in one severe subjective pain in the præcordia and the epigastrium, without any tenderness.

It is not easy to explain why, with no difference in the physical signs, the pain and tenderness should be extreme in some cases and entirely absent in others. It may be that in those with pain there is involvement of the superficial structures, especially the muscles, analogous to that described by Coplin as accompanying pleurisy and pneumonia.

3. The Apex Beat. In the fibrinous form the apex impulse is diffuse, indistinct, and sometimes undulatory, and can usually be appreciated better by touch than by sight. In effusions of any size, it is commonly absent; but a more or less vigorous apex beat *is entirely compatible with the presence of a large effusion*, as was strikingly shown in a patient that came under my observation at the Polyclinic Hospital. Even when the apex beat cannot be seen, it may often be felt as a distinct shock—a point of importance in differentiating between dilatation and effusion. When the apex beat can be located it is found above the lower level of dulness.

4. Pericardial Effusion. Few conditions are more frequently overlooked than effusion into the pericardial sac. At times the signs are unmistakable, even upon very superficial examination; at other times strong doubt encompasses the diagnosis. The signs to which value should be attached are:

1. The character of the right border of the heart, which becomes convex.

2. A change in the cardiohepatic angle, which, from having been a right angle, becomes an obtuse one.

3. As a corollary, the presence of Rotch's sign, dulness in the right fifth interspace, close to the sternum.
4. An increase in dulness upward as far as the second interspace.
5. Flatness with intense resistance on percussion.
6. The position of the apex beat with relation to the lower boundary of the precordial dulness.
7. The shape of the precordial dulness, which is triangular, or, rather, shaped like a trapezium, with the base downward.
8. Bamberger's sign, a patch of dulness with bronchial breathing near the angle of the left scapula.
9. Ewart's sign, a separation of the first rib from the clavicle, making it possible to feel the former throughout its length.
10. The presence of fluid on exploratory puncture.

The best place for *exploratory puncture* and also for aspiration is still unsettled. There is certainly no one point of election suitable for all cases. When the presence of fluid is suspected and the friction sound is still present over limited areas, it is a good and self-evident rule not to make the puncture where friction is heard; nor should it be made at a point where the heart sounds are very distinct. Another wise maxim is to go in slowly and to arrest the needle as soon as the heart is felt to rub against its point. If the apex can be shown by auscultation—or perhaps by palpation—to be above the level of the fluid it will naturally be perfectly safe to tap below it. This will usually bring the point of puncture in the sixth interspace, about the nipple line. At other times the fourth or fifth interspace on the right may with advantage be selected.

The presence of fluid may be simulated by dilatation, and differentiation of the two conditions is far more difficult than the parallel-column method of text-books indicates. I have mistakenly tapped in a case of this kind, and others have had a similar experience. The position of the apex beat with relation to the lower boundary of the heart is of value in the diagnosis; also the presence of a decided cardiac impulse, especially a pronounced diastolic shock. Search should also be made for Bamberger's sign, referred to above.

Great diagnostic difficulties must arise when the fluid occupies an abnormal position; when, for instance, it is collected on the left side of the heart, in which case the precordial dulness extends into the left axilla and simulates pleural effusion. If the fluid is in the pericardial sac, the dulness rarely extends below the eighth rib in the axillary line. Sometimes only tapping can decide the question. When a pleural effusion is co-existent—and this is not at all uncommon—the discovery of the pericardial effusion is rendered far more difficult. When there is double pleural effusion, the pericardial effusion is usually overlooked.[1]

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[1] In one of Thayer's cases, just published (Johns Hopkins Hospital Bulletin, May, 1904), the fluid (over 1200 c.c.) was lying posteriorly to the heart and at the lateral angles of the pericardial sac.

Sometimes pericardial effusions shift with change of posture, especially when the patient is turned to the right. Fluctuation is said to occur at times, but in the cases that I have seen it was not noticed.[1]

Pericardial effusion sometimes simulates *mediastinal tumor.* This is especially the case when the effusion is of long standing. In both conditions there may be dyspnœa, orthopnœa, engorgement of the jugular veins, intense cyanosis, dysphagia, enlargement of the veins of the chest, and extreme flatness on percussion. As at times there is no history suggesting an antecedent pericarditis, suspicion of a mediastinal tumor in these circumstances becomes not unreasonable. A year or two ago I reported a case of this character to the Section in Medicine of the College of Physicians. The patient had the signs that I have mentioned. The cyanosis and dyspnœa were very marked, and the flatness on percussion was most striking. The onset had been insidious, and there was no history of any previous illness. The man was not bedfast, but was able to walk to the Polyclinic Hospital on the clinic days. I took the view that the case was one of either pericardial effusion or mediastinal tumor, although the latter seemed the more likely; but the man died a few weeks later of exhaustion, and the autopsy showed hæmopericardium, the origin of which was not revealed. It is a curious fact that in another case in which the diagnosis of mediastinal growth was for a time entertained, hæmopericardium was also found.

Pericarditis with effusion sometimes simulates *pneumonia.* The distended sac compresses the left lung, and examination of the latter may show impaired resonance and a patch of bronchial breathing in the back. The existence of fever, cough, leukocytosis, dyspnœa, and cyanosis strengthens the suspicion of pneumonia. In a case of this kind that came under my observation at the Philadelphia Hospital, the cyanosis and general prostration were out of proportion to the extent of lung apparently involved. Such disproportion may constitute a point of diagnostic significance.

It is well known that morbid conditions in the chest may cause *pain to be referred to the abdomen,* and give rise to the belief that the disease is located below the diaphragm. I need mention only the so-called "abdominal" pneumonia, which is not rare in children and is also seen in adults. This may resemble appendicitis or perforation of the intestine. Less well known is the fact that pericarditis may cause pain of such severity in the abdomen as to simulate peritonitis.

On the other hand, the interesting observations of Ortner (*Wien. klin. Woch.*, June 29, 1899) teach us that in some cases of peritonitis, chiefly of the hepatic and epigastric regions, a friction sound may be heard over the præcordia, in the total absence of inflammation of the pericardial sac.

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[1] In pericardial effusion, just as in hypertrophy of the heart, skodaic tympany may sometimes be detected over the upper part of the left lung.

A peculiar case, probably belonging to the class described by Ortner, came under my observation at the Polyclinic Hospital in February. A woman, fifty-two years of age, was admitted with severe epigastric pain, tenderness, vomiting, slight jaundice, fever, and leukocytosis. There were no cardiac symptoms, but examination showed the presence of a pronounced friction sound, which was audible from February 3d to February 6th. A few days later a friction sound was discovered in the region of the gall-bladder, extending thence for a short distance upward over the liver. Within a few days this also had disappeared, and the patient left the hospital apparently well.

The only way in which one can escape the error of diagnosing abdominal disease when the trouble is in the chest, and *vice versa,* is by making a careful examination of the thoracic and abdominal organs in all cases.

Even in the early stages of pericarditis signs of *pulmonary congestion* are common, especially on the left side, in the lower axillary region. In three of my cases a distinct generalized *bronchitis* was found.

Fever is, as a rule, present in acute pericarditis, but it has nothing distinctive. When the effusion is purulent the fever may be intermittent, even when the quantity of pus is small. The fever in pericarditis may be present for several days before the friction sound is discovered. Until then the case is usually considered to be one of tuberculosis, sepsis, or typhoid fever. I have thought that in such circumstances the pericarditis probably begins posteriorly, and that the sudden appearance of the friction sound, several days after the onset of fever, is due merely to the fact that the inflammation has extended sufficiently far forward for the friction to become audible.

A curious phenomenon not rarely observed in pericarditis is pronounced *delirium* or other mental disturbance. Da Costa, Balfour, and Musser have made reference to this feature. In one case studied by me with the late Dr. Packard at the Philadelphia Hospital, there was maniacal delirium, which might readily have led an observer less thorough than Dr. Packard astray. Da Costa, in one of his last papers, advised that in every case of acute mania the heart should be examined for pericarditis. The duration of the delirium is variable. In a case of mine, one of tuberculous pericarditis, it extended over a number of weeks and was of a mild character. In his lucid intervals the patient was extremely irritable.

It is difficult to explain the variability in the general reaction of the system to inflammation of the serous membranes. We are accustomed to consider the three serous sacs identical; but pathology proves them to be different, even if histology does not.

The pleural cavity may contain pus for days, weeks, or months without the production of a serious general infection. The peritoneum is a much less efficient guardian of the organism, and suppurative inflammations, as a rule, very speedily influence the condition of the system at large. Acute inflammations of the pericardial sac, whether purulent or serofibrinous, seem to be rapidly productive of constitutional symptoms.

In order to solve these enigmas, we must know:

1. The conditions of lymphatic absorption, both as to direction and as to rapidity.
2. The efficiency of the lymph-gland outposts.
3. The richness in nerve supply, upon which the degree of functional disturbance will depend.

Adherent pericardium more often, probably, than any other important condition comes to the autopsy table undiagnosed. One reason for this is that at times the characteristic signs are altogether wanting. At others, some or all of them are present but are overlooked. Briefly stated these signs are as follows:

1. Systolic retraction of the intercostal spaces.
2. Retraction of the lower portions of the chest in the lateral and posterior aspects—the so-called Broadbent's sign.
3. Diastolic shock or rebound.
4. Absent respiratory movement in the epigastric triangle. The last three are found especially in cases of mediastinopericarditis.
5. Pulsus paradoxus—the so-called Kussmaul's sign—the pulse becoming weaker during inspiration. This is not of much diagnostic value, except when accompanied by inspiratory swelling of the veins of the neck.
6. Diastolic collapse of the veins of the neck—Friedreich's sign.
7. Marked undulation of the heart beat.
8. Early ascites, preceding œdema of the legs.
9. Marked irritability of temper. I have been struck with this, but cannot say that it is either a constant or an important symptom.
10. In rare cases, mild delirium.

For practical purposes the following statements may be made with regard to adherent pericardium. It may be suspected:

1. In cases in which a friction sound has been heard over a great extent of the præcordia and has disappeared.
2. As a probability, in children having a chronic valvular lesion and marked hypertrophy of the heart. In such cases adherent pericardium is commonly found at autopsy. The extreme stretching of the sac and the consequent pressure of the two layers against each other cause alterations of the endothelium favoring a low-grade infection.
3. In adults and children with chronic valvular disease, in whom, with heart sounds of good volume and an impulse of great force, there develop ascites and dropsy of the limbs. In such cases we

may infer that there is something more than the mere valvular lesion. The ancillary factor may not always be adherent pericardium; it may be a fibroid myocarditis, but it is often the former.

4. In the cases of so-called quiet ascites, chiefly occurring in young subjects, in which cirrhosis of the liver and tuberculous peritonitis can be excluded. I have reference to the so-called pericarditic pseudocirrhosis, a subject ably discussed before this society by Dr. Kelly.

Since the beginning of the year to the present time, 9 cases of acute pericarditis have come under my observation: 2 I saw through the courtesy of my colleague, Dr. Thomas G. Ashton, at the Philadelphia Hospital; 2 occurred in my own wards in that institution; 3 were observed during my term of service at the Polyclinic Hospital; 1 was in a patient of Dr. Francis T. Stewart, and 1 I saw in consultation with Dr. L. Podolski. Of these 9 cases 3 were in the female and 6 in the male sex; 4 of the patients were colored persons; 3 were tapped—1 with a negative result.

Etiologically the cases may be classified as follows: Probably of rheumatic origin, 4; associated with pneumonia and nephritis, 1; secondary to operation for the radical cure of hernia, 1; secondary to gallstone colic, 1; of unknown origin, 2. Of the 9 cases, 7 were associated with endocarditis and 2 were unaccompanied with any evidence of endocardial lesion; 2 ended fatally, the autopsy showing fibrinous pericarditis with adhesions.

I have confined myself to a mention of these few details in regard to the cases so as not to lengthen the paper unduly. Those interested in the subject will find these cases more fully discussed in the forthcoming volume of the *Transactions of the College of Physicians of Philadelphia,* 1904, vol. xxvi.

TWO CASES OF PARACENTESIS OF THE PERICARDIUM.[1]

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The glory of modern surgery can be appreciated only by practitioners who, in their youth, were familiar with the almost universal occurrence of operative sepsis. Surgical timidity, due to "inflammatory fever" and its results, made the experienced surgeons of our youthful days skeptical of the opinions of those of us who saw the coming of the new era.

The contention that trephining was not a serious procedure and ought to be adopted in nearly all fractures of the cranial vault[2]

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[1] Read before the College of Physicians of Philadelphia, May 4, 1904.

[2] Roberts, Operative Surgery of Human Brain, Transactions of the American Surgical Association, 1885.